variedly mixed services—medical, surgical, gynecological, and numerous dispensary departments—gives a fair idea of the average occurrence of albuminuria in practice. We would emphasize the frequency of non-renal albuminuria and the occurrence of nucleo-albuminuria alone in a large percentage of cases; and would recommend the addition of saturated salt solution with subsequent heating and acidifying with acetic acid as the safest test for serum-albumin. In practice, the separation of the albumin bodies is unnecessary.

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THE PATHOLOGY AND TREATMENT OF NEPHRITIS.

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The kidney stands in very intimate relation to all the other organs of the body and has a twofold function of excretion and secretion. Placed as it is, at the point where the metabolic products of the system may be thrown off or held back according to their nature and their properties of being useful or useless in the further activities of the body, the kidney, with unerring accuracy, quickly excretes those substances which have outlived their usefulness in the economy and holds back those which have further value. In other words, this organ, being the osmo-regulator of the blood and, consequently, the regulator of the composition and concentration of this tissue, shows almost intelligent power of excretion.

It is worth while bearing in mind the fact that an exceedingly close relationship exists between blood supply and functional activity, an instance of which is the numerous capillary tufts shoved into the beginning of the tortuous secreting tubules. An additional point of value, however, is that the afferent artery, which breaks up into the capillary tuft, has a greater caliber than the efferent vein leading from the tuft. It is, therefore, easier for blood to reach the glomerulus than to leave it, inasmuch as the pressure in the capillary tuft is greater than in ordinary capillaries owing to the resistance in the narrow efferent vein and its capillary plexus. This double capillary supply, both on entering and on leaving the glomerulus, seems to suggest the idea that the highly organized part of the renal tubules is protected from excessive blood pressure by the interposition of the glomerulus as a block.

I shall not discuss the facts, pro and con, leading to the assumption of our present theory of renal secretion further than to state that the water, salts, and probably the pathological sugar and albumin are excreted by the glomerulus, while urea, uric acid, creatinin, with some water and salts, are excreted by the selective activity of the epithelium of the tubules, certain portions of the tubules having different functions in this regard. Naturally, in organic lesions of the kidney disorders of both secreting centres arise, and we have a resulting change in the composition of the secretion. The rate of secretion, necessarily, rises and falls with the blood pressure and also with the velocity of the current passing through the vessels, being influenced, of course, by the activity of the renal epithelium. From the experimental data at hand we must conclude that the process of secretion in the kidney is one of selective activity and not of pure physical filtration.

MODERN CONCEPTIONS OF NEPHRITIS. Normally albumin does not appear in the urine in sufficiently large amounts to respond to our clinical tests. Its presence, clinically, is said to be indicative of a lesion in the glomerular epithelium, as is indicated by the cases of acute interstitial nephritis without albuminuria described by Councilman and others.

It is a very difficult and even impossible task to draw the line between truly physiological or slightly pathological conditions, and those which are clinically characteristic of nephritis. As Stengel says, "The more accurate urinary examinations and the more careful medical diagnoses of recent years have established the fact that it is necessary to distinguish between nephritis in the sense of the pathologist and nephritis or Bright's disease in the clinical sense." The physiological, cyclical, adolescent, orthostatic, and other varieties of albuminuria are rarely, if ever, accompanied by the urinary findings said to be characteristic of nephritis. Febrile albuminuria, on the other hand, is often accompanied, according to von Leyden, von Leube, Senator, and others, by all the elements found in true renal inflammation. We must, therefore, assume that inflammatory lesions in the pathological sense may be present without causing any lasting results, provided these changes have not seriously hampered the functional activity of the kidney. I quote from Stengel: "The presence of slight intertubular exudation of moderate or even extensive, if uncomplicated, degeneration of the epithelium of the tubules, or of slight alterations in the glomeruli, would not justify a positive assertion that the case was one of Bright's disease in the clinical sense. In all of the cases now in mind, albuminuria, the great criterion of nephritis, is likely to be encountered, and yet the pathological conditions are frequently neither serious at the time nor lasting and progressive in character."

The point of all this is, it seems to me, that we must now consider nephritis simply as a renal manifestation of constitutional or metabolic processes. The clinical picture of nephritis may follow these preliminary disorders, depending on the nature of the irritation of the toxin, the time of its action, and the resistance of the kidney to its action. In other words, nephritis is, in the pathological sense at least, due to a direct irritation of the renal parenchyma by poisons, whether they be exogenous or endogenous in character, which have reached the kidney through the circulation, and which exert their untoward influence on the secreting organ. That direct irritation of the kidneys by bacteria is possible we must grant, although, in this condition, we must assume that the lesions are more focal than when caused by the products of bacterial activity. As Councilman says: "The most easily understood lesions of the kidney are focal in character and due to the presence of bacteria in the tissue. Bacteria often pass through the two capillary systems and lodge in the veins of the pyramids. This is particularly the case in hematogenous infections by Staphylococcus aureus. In this connection comes the question of the passage of bacteria through the intact epithelium. Experimentally this has not been shown to take place. It is true that in certain of the infectious diseases, notably typhoid fever, bacteria are found in the urine, but it has not been shown that the kidneys, in these cases, are intact or that the bacteria come from the kidneys."

In a paper recently read before the Chicago Medical Society, Dr. Brewer, of New York, cited several clinical cases, as well as many original experiments, showing a unilateral multiple abscess formation in the kidney following hematogenous infection by various pathogenic and pyogenic bacteria. Clinically, such cases are manifested by acute gastric symptoms with diffuse abdominal pain, later becoming localized in the flanks, with sudden rise in temperature to 105° F. or more, and accompanied by all of the urinary findings of acute nephritis. Such cases must put us on our guard as to the proper diagnosis of inflammations and infections of the kidneys, inasmuch as the treatment, in the latter cases, seems necessarily surgical. We must remember that not all infectious fevers show nephritis as a common complication, but they may, provided the toxin produced by the metabolic activity of the organism in question, is of sufficient concentration and virulence. Also, in the light of Dr. Brewer's observations, we must consider surgical interference in cases showing the symptom-complex mentioned.

When one considers the etiology of nephritis he finds, in every case, that the causative factor is one which has produced marked metabolic or functional disorders resulting in the production of toxins, irritant poisons, changes in the properties of the blood, and other general pathological alterations, which, in themselves, cause irritative and later inflammatory lesions in the kidneys, resulting in what we call, clinically, nephritis. It must be insisted upon that the nephritis will be of a type distinctly pathological or clinical, depending on factors mentioned above. It may still be nephritis in the pathological sense, without being recognizable clinically.

Owing to this fact, one may not venture a classification of the various types of nephritis without, necessarily, leaning toward the one or the other viewpoint. I shall, therefore, simply treat the subject from the clinical standpoint, leaving out of the consideration the well-established pathological fact that we have types of acute parenchymatous, interstitial, and diffuse nephritis. I fortify my statement that we have the pathological type of acute interstitial nephritis without clinical manifestations, by quoting from Councilman: "The acute interstitial non-suppurative nephritis is found in acute infectious diseases, notably in the acute exanthemata, and consists in the presence in the bloodvessels and interstitial tissue of the kidney of cells belonging to the lymphoid series, which are formed in the bone-marrow, spleen, and lymph nodes, and which are brought to the kidneys by the blood. They accumulate in the renal veins, pass from these into the interstitial tissue, and proliferate actively in both places. Unless the cell accumulation is so great as to interfere with the circulation or nutrition they need not give rise to any interference with function, though usually degenerative changes in the kidney due to the toxins of the disease accompany the interstitial changes. These cells never accumulate in the capillaries of the glomerulus, but are found to a slight extent in the capillaries of the cortex, and chiefly in the veins of the pyramids."

Likewise, in considering the chronic type, care must be taken not to confuse the pathological and the clinical standpoints. The interstitial changes observed in chronic cases are, necessarily, secondary to the parenchymatous if pathology be considered, yet, clinically, we do not find all cases of chronic interstitial nephritis as resultants of the chronic parenchymatous type. We must regard the chronic interstitial type as a phase of a general metabolic disorder manifested through the vascular system.

INFLUENCE OF CARDIOVASCULAR CHANGES. As is well known, nephritis, depending on its type, may lead to marked changes throughout the cardiovascular system, as well as be the result of such changes. We cannot explain the vascular changes in the same way for the various types. It is self-evident, it seems to me, that the causes leading to a dilatation of the right heart in acute nephritis must be different from those bringing about a hypertrophy of the left heart with secondary dilatation in chronic nephritis.

In the acute forms we must look for our cause in the increased resistance offered through the accumulation of the volume of blood in the vessels. Water, as is well known, is, perhaps, the most difficult substance to excrete in acute nephritis, as evidenced by the marked oliguria or even anuria observed. In consequence of this retention of fluid the right heart must dilate to accommodate the increased amount, and secondarily the left heart hypertrophies in its effort to force the increased load through the system. Depending entirely on how long this increased heart action is kept up without relief, the result will be sooner or later a breaking down of the compensation and we will then have to deal with a case of heart disease. It is to be said that very rarely does such a sequence present itself in acute nephritis, but it may, as many clinical histories will show. Such clinical pictures are best seen in those acute cases with slight œdema and almost complete anuria. The chronic types are much more often accompanied by this sequence of events.

When we consider the chronic forms of nephritis we see that, although the cause may be different in each type, the result is the same, namely, a marked increase in arterial tension. While in the chronic parenchymatous type the retained metabolic products exert an irritative vasoconstricting effect on the vessels, and at the same time stimulate the heart musculature, the vessels in this type become more permeable and œdema results. Later, the left heart, particularly the ventricle, hypertrophies, owing to the twofold cause of stimulation and increased resistance.

In the primary chronic interstitial nephritis the pressure in the arterial system is high and the contraction of the vessels is great, although the amount of urine is increased. However, the urine in this chronic interstitial form is increased only as regards the water and the easily soluble urinary constituents. In the contracted kidney following arteriosclerosis there is marked interference with the circulation in consequence of loss of tone of the vessels, with resulting increased arterial pressure, and in consequence of the injurious effect of the metabolic products of the blood.

The cardiovascular changes in all forms of nephritis may become so marked that they give rise to the symptoms requiring our greatest efforts, the kidney lesion then becoming of secondary importance from the therapeutic standpoint.

ALBUMINURIA AND CYLINDRURIA. As will be granted from what has preceded, albuminuria is a very common finding in conditions which are of general nature and which give rise to nephritis of the pathological rather than of the clinical form. It is an every-day occurrence to find albuminuria accompanying febrile conditions, yet no signs of clinical nephritis appear to settle the diagnosis. It may be stated as a dogma that albuminuria, *per se*, is not necessarily indicative of nephritis in the clinical sense. Yet, judging from our knowledge of pathology, some lesion must be present in the kidney to permit of such a condition. It is quite possible that the so-called alimentary albuminuria is accompanied by some irritative, though slight, lesion in the kidney, resulting in the removal of the albuminous substances from the blood, yet such substances are, to all intents and purposes, foreign to the blood and may simply be excreted as such.

Albumin is usually considered as excreted through the glomeruli, but, in addition, it may pass from the interstitial bloodvessels and lymphvessels into the urinary tubules, so that, primarily, we may conceive of a glomerulitis whenever albuminuria exists. Either disturbances of circulation, irritation of the parenchyma of the kidney, or changes in the blood may be held responsible for the pathological albuminuria. Space will not permit of a discussion of the theories of Semmola, Lepine, and Rosenbach. It seems to me that it is not improbable that we may, by diligent research, show that differences exist in the albuminuria from various causes. I mean by this that we may find, and certain evidence from experimental work seems to strengthen the assumption, that the albumin excreted in pneumonia, for instance, is different chemically from that observed in nephritis following exposure or excesses. Whether this is or is not the case will be shown, I believe, by the application of the principles so well worked out by Emil Fischer. It is not inconceivable that the various etiological factors of nephritis, particularly the toxic products of bacterial growth, may, in their formation, cause the disintegration and resynthesis of the serum albumin into proteid bodies with different arrangement and different concentration of the various amino-acids, of which they so largely consist. Such bodies would give our clinical tests for albumin and would not be distinguishable from serum-albumin from the clinical point of view.

Regarding cylindruria, it is to be said that here, also, we must be on our guard lest we err in our diagnosis of nephritis. Casts of all kinds appear in the urine in cases showing no clinical manifestations of nephritis, yet we must assume, if our ideas of the origin of casts are correct, that a lesion of the kidney exists. As Emerson says, "Casts alone are no index of the anatomical renal condition. Their most brilliant display is in non-nephritic conditions; the most serious conditions in nephritis may be accompanied by few or none. The more normal the cells the better do they seem to form casts when disturbed: in chronic conditions the cells seem to become accustomed to their condition and to form few casts or none."

CHLORIDE RETENTION. Since the publication of Widal and Javal in 1903 a large number of French and a few German workers have busied themselves with showing that sodium chloride is, contrary to our well-established ideas, a decidedly harmful substance in cases of nephritis. Although Widal and Javal, Achard, Loeper, Claude, Bohne, Castaigne, and others did not originally apply their idea to any but the parenchymatous variety of nephritis, later workers, such as Achard and Paisseau, Brandenstein, Calabrese, Chauffard, Olmer and Audibert, Merklen, Lesne, Vaquez and Laubry, Strauss, and others have extended this original idea to interstitial nephritis, cardiac and hepatic dropsy, epilepsy, etc. These workers have found that the chloride is retained in the system, that concurrently the œdema is increased, and a greater albuminuria is noted. Some recent work of Miller has shown that there is quite as much retention in normal individuals as in nephritics. That œdema is not observed in the normal goes without saying. Ferrannini has shown that although chlorides are retained in nephritis and albuminuria is increased, the general betterment of the condition warrants the use of this substance as a subcutaneous injection. Rumpf, in a recent article, shows that the idea of retention of sodium chloride as an etiological factor in the causation of the œdema of nephritis, is not well grounded. Richter confirms this and argues that sodium chloride is no more responsible for the œdema than are many other factors, especially the amount of water intake. Marischler shows that the kidney of parenchymatous nephritis is permeable for sodium chloride and that the retention is explained by the primary retention of water. Kövesi and Roth-Schulz confirm this idea, and go farther into the physical explanation of the œdema. We observe, therefore, that this assumption of the causal relation of retention of chlorides to the œdema is debatable.

It may be well to ask, How does sodium chloride act in the normal and how in the nephritic metabolism? When NaCl is administered by the mouth it, of course, is absorbed from the stomach and bowel. The blood becomes more concentrated; hence, water passes from the tissues into the blood making this hydremic. The excess of water and of salt is normally excreted by the kidney, since this organ is the osmo-regulator of the blood. NaCl acts, therefore, just as any other salt, such as the citrate or acetate would act, as a diuretic, owing simply to its "salt action." The question might be asked in this connection, Why normal individuals show a retention of chloride? The answer must be rather vague in the present state of our knowledge. The amount of NaCl in the blood, according to Hammarsten, is 6 per 1000, and this amount remains almost constant. With food containing an excess of NaCl, this excess is quickly eliminated, and with food poor in chlorides the amount in the blood first decreases but later increases, after taking chlorides from the tissues. The secretion by the urine is thereby diminished and apparent retention occurs. This retention, it must be insisted on, is only apparent and vanishes as soon as the proper osmotic equilibrium has been established between tissues and blood. According to Marie there are two stages in the retention of chlorides, and these stages depend, of course, on the laws of osmotic pressure and of chemical equilibrium. The first stage of retention, as Marie puts it, is that of fixed chloride (tissue), namely, the combination of the NaCl with the proteids of tissues, which assumption has been experimentally shown to be possible by van Bemmelen, but is doubted by Bugarsky and Liebermann. The second stage is that of circulating (interstitial) chloride. This added point shows us how in the first stage we get a retention, if the NaCl of the blood is insufficient, owing to a poor intake, and is then increased by food rich in NaCl.

If now the kidney is "insufficient," or, in other words, is unable, owing to the inflammatory processes affecting its excretory activity, to excrete the excess fluid and its constituents, there occurs a retention of salt in the circulatory system, a hydremia, and at the same time a plethora, vascular pressure is increased, transudation through the capillaries follows, an attempt is made to adjust the osmotic pressure of the blood and the tissue fluids, salts pass into the tissues, which, in time, become hypertonic, and, in consequence, water follows the salts into the tissues, and œdema occurs as a combined result of increased vascular pressure with transudation and the retention of salts in the tissues. This theory of œdema as a result of osmotic exchange between the blood and tissues was advanced by Loeb in 1898, and has been reached by the French school, especially by Widal and Javal, working from different standpoints.

It is difficult to say just why nephritics show increased albuminuria after increased intake of NaCl, yet one may call to mind the results of Hoppe-Seyler, von Wittich, and Nasse, showing that an albuminous solution filters stronger the greater its content of salt. This purely chemical fact may also be applicable, as far as the purely physical theory of filtration through the kidney is concerned. On the other hand, one may assume that the kidney has become more permeable for albumin, or that the albumin itself has become more diffusible, inasmuch as various workers have shown that normal individuals, in time, show an albuminuria under the influence of increased intake of NaCl. There are difficulties in the way of both explanations. In the first place, if the kidney has become permeable for albumin, then would it not be permeable, at the same time, for the increased chlorides? If the albumin is more diffusible, it is not as diffusible as the NaCl, and yet the latter remains and the former passes out. We must again revert to the osmotic theory for our explanation. Proteids possess only a slight osmotic value, while the salts have very large equivalents. The osmotic and the diffusion currents extract the fluid and the salts first from the tissues and then later from the blood back into the tissues. The proteids are not affected by this interchange, unless we may assume, with Semmola, that some change in the constitution of the proteids, by which they become more diffusible, is brought about in nephritis.

In this connection, I wish to mention some experiments which I began under Senator and have continued at the Cook County Hospital in Chicago. It was found that the urine of nephritics showed the separation of a coagulatory inhibitory substance and at the same time of a distinct ferment with marked peptic characteristics. This latter was in all probability simply an increased amount of the normal peptic ferment found in the urine by Grober, Gehrig, Grützner, Mathes, Rosenberg, Stadelmann, and others. This separation of a substance in the urine which favored or inhibited coagulation of proteid in pneumonia has been previously worked out by Lenobel, Kun, and Lochbihler. It is possible that the albumin becomes more diffusible solely through the presence of these two substances whose identity I am at present unable to disclose. It is, however, a very strange fact that NaCl will increase or cause an albuminuria. The explanation best suited, it seems to me, is that we have a pure toxic albuminuria. This toxic action of NaCl is well known physiologically from the work of Loeb, Moore, Neilson, and others.

Œdema. I shall refer in this connection only to the œdema caused by the lesions of the kidney resulting in certain pathological changes of the metabolism of the fluids and solids of the blood and tissues. I exclude a discussion of cardiac and hepatic œdema as well as of "hydrops cachecticus," which have no direct place here, although I am convinced of the correctness of the reasoning of Kövesi and Roth-Schulz that the cause of cardiac and renal œdema is a common one.

We must abandon the old conception of Bright's that œdema is caused by hydremia of the blood resulting from the loss of albumin, in consequence of which the watery constituents more easily passed out of the vessels, just as we must give up Bartels' and Stewart's ideas of a hydremic plethora, because we can show that retention of urine, *per se*, will not bring about an œdema. Cohnheim assumes an inflammatory change in the vessels by which they become abnormally permeable. Senator remarks: "Under the influence of certain injurious properties of the poison circulating in the blood, the capillaries of the glomerulus suffer first and then, if the injurious action be strong enough and long enough continued, the interstitial and other vascular areas outside of the kidney, such as those of the skin and serous cavities, become affected. A glomerular affection is always to be looked for when œdema is present, but not vice versa."

Kövesi and Roth-Schulz would make the insufficiency of the kidney in itself responsible for the changes resulting in œdema. The changes in the kidney excite abnormal metabolic processes by increasing the watery and solid constituents of the blood and preventing the elimination of the poisonous toxic and autolytic products. As a result of increased vascular pressure combined with abnormal permeability of the capillary walls and increased osmotic change œdema follows. It is well to remember in this connection the role played by the perspiration, both sensible and insensible. As Bidder and Schmidt, Seegen, and others have shown, the amount of perspiration depends on the temperature and the degree of saturation of the surrounding air. However, it is a well-known fact that nephritics perspire very slightly. von Koryani comes forward with an explanation of increased osmotic pressure of the blood, raising the point of evaporation of fluid, and, hence, decreasing the perspiration. It is possible thus to explain a part of the effect of NaCl in increasing the œdema by lessening the loss of water by the skin and lungs. The amount of fluid taken in is also a large factor in the causation of the œdema. As the fluid is absorbed just as it is normally, the hydremic condition is increased, and with the resulting insufficiency of the kidney, water elimination being the first abnormal phase noticed when the kidney becomes affected, aggravation of an œdematous condition follows. There is thus a distinct reason for limiting the intake rather than increasing it, as we cannot flush the kidney in this way owing to its lessened excretory activity. The result is an overburdening of an already highly taxed heart and an increase of the œdema.

Uremia. In this discussion, up to this point, I have not attempted to take up the effects of nephritis on the system in general. Under the head of uremia one must bring in certain points of general pathology and pathological chemistry in an attempt to show that this condition is a result of purely metabolic processes. As œdema is a combined result of renal and vascular causes on the metabolism, so is uremia the result of changes on the cerebral centres and on the digestive system, due to disturbances in general metabolism, together with insufficient kidneys. The subject of autointoxication is one of general interest whether it be enteric, hepatic, or renal.

The theories advanced as to the causation of uremia have for the most part as a basis the idea of overloading of the blood with urinary constituents as a result of insufficient kidneys. Opposed to these theories are those that hold that hydremia with resulting œdema of the brain is at the bottom of the symptomatology. These latter ideas are especially advocated by Rees and Traube, but do not seem to be convincing, inasmuch as certain factors assumed by the advocates are at times missing and hence cannot be regarded as causative of all cases. I refer here, of course, to the hypertrophy of the heart and to the brain œdema. We must, however, grant that the œdema, when present, may be responsible for certain nuclear symptoms observed in uremic attacks.

Space will not permit an enumeration of all the factors advanced in support of the chemical theory as opposed to the physical. We all know that the retention of almost every constituent of the urine has, at one time or another, been held to be as the causative factor in uremia. One by one these substances have been abandoned until now we cannot grant that any one substance is specific in the causation of this group of symptoms. From the time that retention of urea as an etiological unit was advanced by Wilson until the list had been exhausted by Bouchard in his assumption of organic urotoxins, we have been obliged to change our ideas of the factors responsible for the trouble under discussion. Opposed to these views are those of Frerichs, Demjankow, and the closely related one of Treitz, which assume an ammoniemia as the causative agent.

Strauss found that ammonia was increased in the blood, but at the same time other nitrogenous bodies (neither albumin nor urea) are present, which must play a role in the etiology of uremia. He shows that while the salt content of the serum in uremia does not change, the amount of the nitrogenous constituents contained in the hydremic serum, that is, the retention nitrogen, is, as a rule, increased to a great extent. In connection with this work of Strauss one must mention the findings of Gumlich and Richter that ammonia, alloxur bodies, and extractives are markedly increased in the urine of uremics. It is unnecessary for me to discuss the relation of an increased ammonia excretion to an increased acid formation in the tissues. This regulation of the metabolism is a well-established fact. This taken with the idea of Strauss gives us a very rational basis for the etiology of uremia. Recently, Croftan, reviewing the facts given above, as well as certain points brought out in his own experience, advances the theory that uremia is primarily dependent on insufficiency of the liver, following a general metabolic disorder, and that the kidney is only secondarily important in the causation. This theory has a certain element of plausibility, owing to the fact that, in just such cases of insufficient liver, disturbances of the urea formation, ammoniemia, and irregular autolysis occur and lead to increased acid formation either of the amino or oxy series. However, were it not for the disturbance of the excretion by the kidney, uremia would not occur. The general metabolism of the proteids is at fault in the theories advocated by Senator and by Croftan. As Croftan says, "The liver is not the only organ affected, and uremia interpreted as above is not so much the result of hepatic derangement as of a general metabolic disturbance."

This view of the terminal syndrome of some forms of nephritis is quite in harmony with our idea of nephritis as primarily a constitutional disorder with secondary involvement of the kidneys.

TREATMENT. In a discussion of the treatment of the various forms of nephritis emphasis must be laid, at the beginning, on the individual factor in the handling of our patients. It is very irrational to order, for instance, an exclusive diet of milk in all cases, just as it is unsound treatment to deprive all of the nephritics of dark meats. Each case is a law unto itself, and, in consequence, I cannot attempt to do more than lay down general principles which may be varied to suit the particular case in hand.

Dietetics. No factor in our handling of a nephritic patient plays a greater role than the dietetic treatment. A rational basis for such treatment has at last been worked out, thanks to von Noorden and others, so that we are today working with surer principles than before.

As a general rule, it may be said that, in the beginning and during the height of an acute nephritis, starvation for four or five days is the best treatment. Of course, this starvation diet need not be absolute in all cases, as for instance in the lighter types which we observe in most fevers; but in the forms following scarlatina, for instance, starvation more or less absolute depending on the intensity of the process, as shown by oliguria and marked œdema, must be practised. It may be stated here that we should not, for reasons previously mentioned, judge too severely from the amount of albumin or the number of casts; but we should rather watch the clinical symptoms closely for signs of general disorder. It is much better practice to follow the œdema and the heart symptoms rather than the urinary in drawing conclusions as to the severity of a special case. As we know, recovery usually follows in cases of acute nephritis if a certain period of danger can be passed through. It is our duty to avert this danger, if possible, and, therefore, starvation is to be forced in cases of greatest severity, limiting the intake of fluid as far as possible, and giving ice to quench thirst.

It is a matter of common knowledge that in acute nephritis water is the substance most difficult to get rid of through the kidneys. How irrational then is the idea of ordering a rigid milk diet and forcing water. The main effect of such treatment will be the overloading of the blood with material that cannot be excreted, and thus increasing an already existing œdema, and, at the same time, causing the heart to work harder to force the increased volume of blood and, in consequence, increasing the danger of hypertrophy and later dilatation of this organ.

In cases which are less severe and are not accompanied by a high degree of oliguria and œdema, milk to which has been added a little calcium carbonate to form the insoluble calcium phosphate may be given in quantities not to exceed, on the average, three pints, and the remaining caloric value necessary to maintain the standards of the system may be made up of fat in the form of cream and cereals. It must be remembered that, in our efforts to protect the kidney from overwork, we must not go so far as to injure other organs and, at the same time, the whole organism. The principle of "Schonungstherapie" has certain natural boundaries and these must not be overstepped. Our idea must be to preserve the general nutrition at as high a standard as is compatible with the best working powers of the kidney. We therefore gauge our diet largely by the degree of oliguria and œdema, decreasing the former as the latter increases, and vice versa. Meat broths should be interdicted to the last, inasmuch as these contain, practically, nothing but uric acid, purin bases, and creatinin, and, in consequence, can have very little nutritive value and may irritate the kidney attempting to excrete them.

In the later stages of acute nephritis the food may be gradually increased as desired, care being taken to avoid a large excess of proteid. As is often the case, three or four quarts of milk are given daily in the convalescent stage. Such treatment is bad because the fluid, finding its way into the circulation, may overburden a weakened heart; furthermore, the amount of proteid thus introduced is unnecessarily high. It may be said that it is unnecessary to limit the patient to white meats by depriving him of the red meats. No difference is found between them as to their influence on the general metabolism and on the regenerative processes in the kidney.

Exactly the same procedure as outlined above should be followed in cases showing acute exacerbations of the chronic form.

When we consider the chronic types of nephritis we have a different viewpoint to study. These types are essentially chronic, that is, they continue for long periods of time. As curing such a case is, at present, out of the question we should direct our efforts to maintaining the general body activity at as high a plane as is consistent with the condition of the kidney. Sooner or later the chronic forms, particularly the interstitial type, become practically heart cases and should be treated as such. We are, therefore, justified in allowing the patient more or less latitude in the choice of his diet, remembering certain precautions. The intake of fluid should be limited to some extent. The idea of forcing a milk diet with plenty of water is harmful not only to the kidney, but, what is more important, to the heart. A liberal proteid diet is permissible and no difference need be made as to white or red meats. Condiments are to be avoided as far as possible. Fresh fruits and vegetables may be ordered as desired, recalling the fact that von Noorden advises against the use of radishes and celery, but permits asparagus. Coffee, tea, alcohol, and tobacco should be used with great moderation.

In regard to dechloruration, it is to be said that the limitation of salt in the diet is unnecessary, since it seems to me that the theory upon which its deprivation is based is not tenable. Experiments are still necessary to prove that the œdema is dependent on the retention of salt and also to prove that the salt is actually instead of apparently retained. It is true, however, that some cases are benefited by dechloruration, but it seems to me doubtful that such results are due entirely to the withdrawal of salt.

It must be recalled that each person needs a certain definite amount of energy to maintain himself in healthy equilibrium. With a nephritic, the same thing is true within certain limits. As the energy comes from the food it is rational to construct the diet with definite caloric values in view, remembering that it is advisable to restrict the fluids to some extent and to confine the proteid intake within 100 grams (on a basis of 70 kilograms of body weight). If the reasoning of Chittenden and Folin is correct we must reconstruct our ideas of the proteid need of the system to a basis far below that of the old-established formula of Voit (118 grams of proteid on a basis of 70 kilograms of weight). An excess of proteid is the dangerous thing in the dietary of a chronic nephritic.

We must not forget in our dietetic treatment of nephritis that the digestive powers of the patient should be carefully watched. The digestive tract is a prolific source of toxemia and should, therefore, be carefully regulated. Likewise, the functional power of the liver should be maintained at a high standard to protect the system against accumulation of abnormal products of a deranged hepatic metabolism.

Hygienic and Physical Therapy. Almost as much importance attaches to these phases of the treatment of nephritis as to the dietetic. It is naturally in the chronic types that we have to do with such measures to the greatest extent. Of course, in the acute forms we resort to sweating and purging processes with the idea of relieving the kidney of unnecessary work and at the same time of controlling the œdema. As these sweating processes are, in some cases, very weakening, it is good practice to stop them at the first sign of weakness of the heart, provided no uremic symptoms are present; if these develop, we must, of course, use all our efforts to control this condition. It is irrational to allow the free use of fluids and then force the sweating process, since both treatments are followed by weakness of the heart.

In chronic types, hydrotherapy plays a helpful role in aiding the elimination of water and salts, but it in no way aids the excretion of the harmful nitrogenous products. Proper clothing, preferably woollen, and bathing are to be ordered, and the patient should be directed to spend as much time as possible in the open air. The drier the air the better, as this aids the evaporation from the skin. Exercise is a very important element in the proper handling of these patients, but should never be indulged in to excess. Exercise tends to strengthen a weakened heart, but if excessive will lead to serious complications, as the heart is necessarily under great strain in all cases of chronic nephritis. Moderation in all things is the keynote of the hygienic and physical treatment of these cases.

Symptomatic Treatment. There are several indications for the treatment of nephritis which may fall under this heading: (1) Maintenance of cardiovascular equilibrium; (2) regulation of the œdema; and (3) regulation of the bowels and of the liver. The first two of these are more or less closely related, inasmuch as the œdema is partially a result of disturbed cardiovascular equilibrium.

The maintenance of the cardiovascular equilibrium is, in most cases, of the greatest importance. As the essential feature of the circulatory change is a high arterial tension with or without œdema, it is readily seen why we should be on our guard in following such cases. The œdema may come from the kidney lesion direct or later from the breaking down of a compensated heart. It is usually only a matter of time until this latter factor enters into the case. It is important to recognize which of these two factors is predominant in the causation of the œdema, as the treatment is somewhat different in each case. In those cases in which the heart is fully compensated, our indication is to regulate the blood pressure within safe bounds, a certain degree of increased pressure being necessary. These cases should always be followed with some instrument to indicate the pressure, as a routine. As stated above, a careful dietetic and hygienic regulation, together with free action of the skin and bowels, is usually adequate in controlling the arterial tension and œdema. If these measures do not prove effective, we must use some vasodilator drug or a combination of such drugs in our efforts to maintain a proper equilibrium. The nitrites of sodium or potassium are often found beneficial. Erythrol tetranitrate or nitroglycerin is often given with this object in view. As a general rule, we should resort to vasodilatation whenever the pressure in the adult male reaches 150 mm. of mercury, and at even lower pressures in the female. Diuretics are unnecessary and do no good in purely renal œdema. We may resort to colonic irrigation, which may prove useful in reducing the arterial tension and œdema.

There is no indication for digitalis in the treatment of nephritis as long as the heart is compensated. However, when signs of failing compensation appear and the œdema becomes one of the cardiorenal type, digitalis is the drug to use. As long as the preparation is trustworthy any form of this drug may be used, although we are taught that the infusion has special merit in such cases. Care should be taken to watch the action of the drug lest its action becomes cumulative and the second and third stages of its action become manifest.

I have spoken already concerning the proper regulation of the bowel and the liver. I need only say here that this indication is very important, since we have in the digestive tract, as well as in the liver, a source of constant danger. Enteric and hepatic toxemias are too common to need discussion.

Tension of the skin from the œdema is to be relieved by puncture under aseptic precautions. Effusions into the various serous cavities are to be handled by puncture or paracentesis, depending on the location.

As the heart becomes more and more weakened, we find that the patient complains of dyspnœa, dizziness, etc., in the reclining posture. In such cases the upright position is best suited to ease these symptoms.

Care should be taken to watch for symptoms of uremia and to give brisk purgatives and diaphoretics as soon as precursors, such as stubborn headache, dizziness, nausea, vomiting, etc., are noted. In view of recent advances it might be advisable to force an alkaline treatment with the view of neutralizing the excessive acids produced. Venesection may be resorted to if the blood pressure gets too high. Subcutaneous and even intravenous saline injections will often aid the system in throwing off the substances which are at the bottom of the trouble

Surgical Treatment. In recent years surgical treatment has been advocated, especially by Edebohls and Ferguson, in the chronic types of nephritis. Briefly stated, the surgical intervention consists in nephrotomy or decapsulation. This procedure leads to an increased elimination by drainage of the accumulated products. As the operation is done in the primary stage of anesthesia, the untoward effect of the anesthetic is very much lessened. The whole operation consumes only three to four minutes, and, judging from case reports, is often very beneficial. Much may be expected of this form of treatment, as it is surely rational in some cases and is successful in many.

In cases such as those described by Brewer, nephrectomy seems to have yielded cures. It may be that, in these forms also, decapsulation with drainage will be useful; it will lessen the danger of operative procedure.

THE USE OF THE X-RAYS IN UNRESOLVED PNEUMONIA.

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That exposure to the x-rays might be useful in the treatment of unresolved pneumonia was suggested by one of us[1] in discussing the effects of the x-rays on metabolism in leukemia. It was then stated that the remarkable effect that this agent usually exerts upon metabolism is most readily and most satisfactorily explained by considering it to be due to action upon ferment processes normally resident in the tissues; the action in this instance apparently being to accelerate autolytic processes inherent in the tissues. The reasons then evident for adhering to this view were given at that time, and continued studies of the effect of x-rays upon metabolism in various diseases—studies now amounting to twelve—have emphasized our opinion that this is the most satisfactory conclusion to be reached. Further consideration will be given this point in a more extended discussion of the accumulated results in the various conditions studied, and it is, therefore, unnecessary to burden the present communication with it.

Granting the correctness of the view mentioned concerning the

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[1] Musser and Edsall. Trans. Assoc. Amer. Phys., 1905; Univ. Penn. Bull., September, 1905.